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Review of coronary subclavian steal syndrome

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ABSTRACT

The clinical benefits of using the left internal mammary artery (LIMA) to bypass the left anterior descending artery are well established making it the most frequently used conduit for coronary artery bypass surgery (CABG). Coronary subclavian steal syndrome (CSSS) occurs during left arm exertion when (1) the LIMA is used during bypass surgery and (2) there is a high grade (\geq 75%) left subclavian artery stenosis or occlusion proximal to the ostia of the LIMA resulting in "stealing" of the myocardial blood supply via retrograde flow up the LIMA graft to maintain left upper extremity perfusion. Although CSSS was once thought to be a rare phenomenon, its prevalence has been underestimated and is becoming increasingly recognized as a serious threat to the success of CABG. Current guidelines are lacking on recommendations for screening of subclavian artery stenosis (SAS) pre- and post-CABG. We hope to provide an algorithm for SAS screening to prevent CSSS in internal mammary artery bypass recipients and review treatment options in the percutaneous era.

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Background

The left internal mammary artery (LIMA) is the preferred and most commonly utilized conduit for myocardial revascularization during coronary artery bypass graft (CABG) when used in situ because of its superior patency rate and survival benefit when grafted to the left anterior descending artery (LAD) as compared to saphenous vein grafts [1–10]. The LIMA's long-term durability has been attributed to its (1) functional endothelium that produces potent vasodilators and inhibitors of platelet function and (2) continuous elastic lamina that inhibits smooth muscle migration and thus arteriosclerosis [11–13]. During CABG, the proximal end of the LIMA is typically left attached to the left subclavian artery while the distal end is harvested and anastomosed to the diseased epicardial coronary artery. Presence of a significant proximal left subclavian artery stenosis (LSAS) can lead to functional LIMA graft failure despite having a disease-free graft by stealing of blood from the myocardium. This phenomenon is known as coronarysubclavian steal syndrome (CSSS) (Fig. 1) and its consequences include angina, acute coronary syndrome, new-onset and decompensated heart failure, and malignant ventricular arrhythmias [14–20]. These serious and potentially catastrophic implications behoove screening for subclavian artery stenosis (SAS) prior to CABG and continued active surveillance for the interval development of SAS post CABG [21,22]. While SAS in a patient with a history of CABG can also result in isolated or concomitant posterior neurological perfusion compromise from the more commonly described subclavian-vertebral steal syndrome (SSS), this review will focus on the prevalence, risk factors, anatomy and physiology. screening and diagnosis of CSSS, and current treatment options in the percutaneous era.

Prevalence and risk factors

The largest registry to date studying the prevalence of SAS (defined as a \geq 15 mmHg interarm brachial systolic pressure difference) found it in approximately 2% of the general population

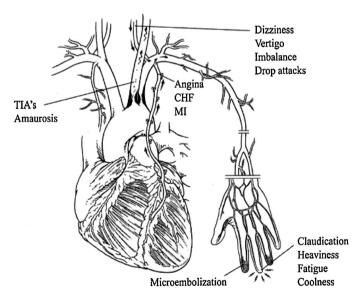


Fig. 1. Clinical consequences of coronary subclavian steal syndrome. A high-grade proximal left subclavian artery stenosis results in reversal of blood flow up the left internal mammary artery away from the heart to maintain perfusion of the left upper extremity. Consequences of coronary subclavian steal syndrome are myocardial ischemia/infarction, heart failure, and malignant arrhythmias. CHF, congestive heart failure; MI, myocardial infarction; TIA, transient ischemic attack. Reproduced, with permission, from Takach et al. [57]

and 7% of a clinical population enriched with patients with known or suspected peripheral artery disease (PAD) [23]. In patients who have both PAD and coronary artery disease requiring CABG, 11.8% were found to have proximal LSAS [24]. However, not all LSAS resulted in CSSS. It has been estimated that CSSS complicates 0.2% to 6.8% of patients who have undergone CABG with a LIMA graft [25].

PAD is the single strongest predictor of SAS with its presence conferring a 5-fold increase risk of having SAS. Other factors associated with SAS include past smoking, current smoking, higher levels of systolic blood pressure (SBP), and lower levels of high-density lipoprotein (HDL) cholesterol [23]. In a series of patients undergoing simultaneous brachiocephalic and coronary angiography, 21% of patients were found to have a significant LSAS defined as a stenosis of more than 50%, complete vessel occlusion, or aneurysm with triple-vessel disease coronary artery disease (CAD) identified as the strongest predictor (OR 9.917; 95% CI 2.2–43.8, p = 0.002) [26].

While CSSS has previously been considered an uncommon complication of CABG surgery, several authors have postulated that this is likely an underestimation as the number of internal mammary artery (IMA) grafts has increased and life expectancy has improved in parallel [24,27].

Etiology, anatomy, and pathophysiology

Atherosclerosis is responsible for more than 90% of SAS. Less common etiologies of SAS include arteritis, inflammation, radiation exposure, compression syndromes, fibromuscular dysplasia, and neurofibromatosis [28]. The proximal portion of the left subclavian artery, defined as the segment that runs medial to the anterior scalene muscle, is three times more susceptible to flowlimiting disease than other supra-aortic vessels. This has been attributed to the acute angle between the origin of the left subclavian artery and the ascending aorta, which has been postulated to increase flow turbulence and thus the potential for atherogenesis [28,29]. A normal luminal diameter of the proximal left subclavian artery is 8-10 mm in an adult male and 7-9 mm in an adult female. The proximal left subclavian artery gives off three important branches: vertebral, internal mammary, and thyrocervical trunk arteries. The costocervical trunk and dorsal scapular artery arises from the middle and distal portions of the subclavian artery, respectively.

CSSS occurs during left arm exertion when (1) the LIMA is used during CABG and (2) there is a high grade (\geq 75%) LSAS or occlusion proximal to the ostia of the LIMA compromising antegrade blood flow to the LIMA graft and also resulting in "stealing" of myocardial blood supply via retrograde flow up the LIMA graft to maintain left upper extremity perfusion, similar to SVSS when the vertebral artery blood flow to the brain is reversed to supply the ipsilateral arm. Another mechanism by which myocardial blood can be stolen from an IMA bypass graft occurs in end-stage renal disease patients receiving hemodialysis through an arterial-venous fistula in the ipsilateral arm [30].

Screening and diagnosis

Both the 2011 ACCF/AHA and 2014 ESC/EACTS guidelines on myocardial revascularization fail to address SAS as a potential threat to the success of CABG and provide guidance on SAS screening pre- or post-CABG with an IMA graft [31,32]. Bilateral blood pressure measurement is the simplest and most costefficient method for screening of SAS, but can miss patients who have equal bilateral subclavian artery stenosis. Subclavian artery angiography remains the gold standard and can be performed during coronary angiography. However, this must be Download English Version:

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